per cubic millimeter. The colloidal gold curve is variable, but in the presence of a negative spinal fluid Wassermann a tabetic or paretic gold curve in a patient with clinical symptoms of encephalitis should be of significance in the diagnosis.

4. The etiology is extremely obscure.

5. The pathology is characterized by perivascular infiltration of round cells in the nuclei of the bulb.

6. The majority of patients recover, but mortality does occur.

A REPORT OF AN EPIDEMIC WITH CERTAIN CASES PRESENTING THE PICTURE OF MENINGO-ENCEPHALITIS.

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Of lethargic encephalitis Achard[1] aptly says, "La maladie est polymorphe et acyclique." Clinically, epidemic encephalitis presents a most interesting group of syndromes which vary widely in their symptomatology. From time to time various forms of this disease have been added to the lethargic type, the first to be described. Syndromes resembling paralysis agitans, catalepsy, chorea, mania, bulbar palsy and other conditions are of such frequent occurrence that the name "epidemic encephalitis" has largely replaced the original term "lethargic encephalitis."

The lesions of this disease are fairly definitely limited to the brain-stem and the basal ganglia and consist of a perivascular infiltration of lymphocytes and plasma cells, engorgement of the vessels and degenerative changes in the ganglionic cells. The pathological changes, being most commonly confined to the brain-stem and basal ganglia, are sufficient to produce most, if not all, of the various syndromes described.

As a rule, however, the meninges have been relatively uninvolved by the pathologic process and examinations of the cerebrospinal fluid have seldom given a cell count that would indicate any marked degree of meningitis.

Hala and Smith[2] reported 1 case with definite signs of meningitis: The cell count in the cerebrospinal fluid was 480 cells per c.mm. and on the thirtieth day after the onset a motile, gram-negative bacillus

[1] Lethargic Encephalitis, Bull. de l'Acad. de méd., Paris, 1920, lxxxiii, 106.

[2] A Case of Meningo-encephalitis (Lethargic Encephalitis), Arch. Neurol. and Psych., 1920, iii, 160.

was found in smears from the spinal fluid. The pathological examination showed a purulent exudate in the sulci between the convolutions and a bacillus similar to the one in the spinal fluid was found present in the exudate. Laboratory animals died three to seven days after inoculation. The authors state that "the cultural characteristics of this organism on sugar coincide with those of no other organism which we know." Their conclusion is that "the bacillus probably belongs to some intermediate class of the colon-typhoid-enteritidis group."

Barker, Cross and Irwin[3] stated that "only in a relatively few cases are the clinical signs of an outspoken meningeal irritation present. In one of our series there was pain in the neck and back, some rigidity of the neck, a positive Brudzinski sign and a positive Kernig sign. That there is a slight patchy involvement of the leptomeninges in the inflammatory process in many cases has been shown, however, by postmortem examinations and by the increased cell count and the positive globulin reactions in the cerebrospinal fluid."

Tilney and Riley[4] found definite pathological changes in the spinal fluid in only 1 case out of 15. A diplostreptococcus was present in the fluid upon incubation, on culture, and in the stained smears. Subsequent tests of the fluid in the same case failed to show any organism to be present.

Bassoe and Hassin[5] noted in one of their cases pial changes indicative of a mild leptomeningitis. The pia-arachnoid was infiltrated and frequently showed distended meshes containing congested hyperemic vessels.

Flexner[6] reported increased cells and globulin in the cerebrospinal fluid but less than in poliomyelitis or meningitis.

Heiman[7] states that there is frequently moderate rigidity of the neck. Of eight spinal fluids examined he found definite changes in only one, which had a cell count of 80 and a positive albumin test.

Bassoe[8] suggests with some hesitation that this virus also may produce a syndrome characterized by meningeal irritation, and by irritation or paralysis referable to the spinal or cranial nerve roots. He reports 2 cases of this type.

[3] On the Epidemic of Acute and Subacute Non-suppurative Inflammations of the Nervous System Prevalent in the United States in 1918–1919; Encephalomyelitis; Polyneuritis and Meningo-encephalomyeloneuritis, Am. Jour. Med. Sc., 1920, clix, 170.

[4] Epidemic Encephalitis, Neurol. Bull., 1919, ii, 106.

[5] A Contribution to the Histopathology of Epidemic (Lethargic) Encephalitis, Arch. Neurol. and Psych., 1919, ii, 24.

[6] Histologic, Pathological and Clinical Facts and Epidemology in Brief, Jour. Am. Med. Assn., March 27, 1920, lxxiv, 865.

[7] Am. Ped. Soc. and Jour. Am. Med. Assn., July 19, 1919.

[8] The Delirious and Meningoradicular Type of Epidemic Encephalitis, Jour. Am. Med. Assn., 1920, lxxiv, 1009.

In a study of the literature with reference to the epidemiology of epidemic encephalitis, we found no reports of any definite contagiousness or of any tendency for the disease to spread through a household.

In the Local Government Board Report[9] it is noted that of 58 cases investigated there was no instance of more than 1 case in the same household and Draper in this report says there was but one instance of more than 1 case in a family.

The following series of cases is of special interest because of the marked meningeal symptoms, the high degree of multiple incidence, the change in type of involvement and course and the bacteriological findings.

The epidemic occurred in a village of 743 people in southwestern Minnesota. There were 11 cases: 5 of which were in two related families; of the other 6 there were 3 in one family and 2 in another family. Six of the 11 patients died. Two of these patients were seen by one of us during their illness. Case No. IX was seen two weeks after the onset and case No. X was examined one week after the onset of the disease.

CASE I.—T. D., male, aged nineteen years, was the first possible case. He had not been in good health since 1916, when he was operated for exophthalmic goiter. His fellow workmen in the local produce company where he was employed had noticed an apparent failure in strength for several weeks before the onset of the acute symptoms.

On December 9, 1919, he came home from work complaining of not feeling well. The following morning he was nauseated and his chief symptom was fatigue. At 10.00 P.M., December 10, he became restless and delirious. A physician, who was called only shortly before death, noted some pulmonary involvement with blood tinged sputum and made a diagnosis of pneumonia. Death occurred at 3.00 A.M. December 11, about twenty-one hours after onset of symptoms.

CASE II.—M. G., male, aged twenty-five years, came to the village December 9 and left for home at noon, December 12. While in the village, he lived with his brother (Case VII) and his brother's wife (Case IX). This brother was the local butcher with a shop next door to the produce company where T. D. (Case I) worked. M. G. became ill on his way home on the train during the night of December 12–13. On the morning of December 13, he was driven from the station to his farm, became unconscious soon after his arrival home and died December 14 at 9.00 P.M. The entire dura-

[9] Report of an Inquiry into an Obscure Disease, Encephalitis Lethargica, 1918. Reports of Local Government Board on Public Health and Medical Subjects, London, New Series No. 121,

tion of illness was about forty-eight hours. The diagnosis in this case was said to have been spotted fever.

CASE III.—J. R. (son of patient No. IV), male, aged ten years, became ill December 12 at 2.00 P.M. and died December 13 at 2.00 P.M. During his illness, he was not restless, but vomited a few hours after the onset of the illness and was delirious during the night. He did not seem to be in great pain or to suffer from distress. There was no Kernig or rigidity of the neck at any time.

CASE IV.—R. R. (mother of patient No. III), female, aged forty-one years, became ill December 13 with severe headache, mostly in the back of the head. The next morning, there was a purpuric rash on body and the temperature was 100.4° and the pulse 80. She got up, however, and walked about the room. On December 15 she complained of headache, backache, vomiting and pains in the limbs. At this time, she stated that she felt well except for the need of a good night's sleep. There was occasional deafness from the second day of illness. She felt drowsy most of the time but would answer questions rationally. There was a slight icteric tinge to the sclera. There was no Kernig or rigidity at any time. The leukocyte count of the blood was 34,300 per c.mm. Death occurred December 18 after a period of partial unconsciousness. The duration of the illness was about five days.

Autopsy was performed December 20, 1919, thirty-eight hours after death. The body was embalmed very shortly after death. The body was that of a well-developed stout woman. There was no edema; there was an icteric tinge to the scleræ of the eyes but this was not demonstrable on the skin. Small petechiæ were seen over the chest, abdomen, arms, hands and thighs. There were a few fresh fibrinous adhesions over the lateral and interlobar surfaces of the right lung. There was no pneumonia found. The spleen weighed 230 grams and showed the gross and microscopic evidences of acute splenitis. The liver showed fatty metamorphosis and chronic passive hyperemia. The kidneys showed moderate cloudy swelling both grossly and microscopically.

The brain showed a fairly large amount of greenish very thick purulent exudate in the pia-arachnoid over both cerebral hemispheres, especially in the sulci. This exudate was also found over the superior surface of the cerebellum. All the meningeal vessels were dilated, especially those on the left side. The lateral ventricles and the third ventricle contained no excess of fluid. Careful coronal sectioning of the brain showed no gross lesions in the brain substance. Smears of the meningeal exudate showed large numbers of polymorphonuclears and a moderate number of mononuclears leukocytes. There were a very few possible intracellular diplococci but one could not be absolutely certain about

these. No microscopic sections were made from the brain substance. The body had been embalmed so that cultural study was impossible.

CASE V.—H. K. (brother of patient No. VIII and nephew of patients Nos. VI and X and all lived in the same house), male, aged five years, became ill during the forenoon of December 18. He began to lie around during the afternoon but did not want to go to bed. In the evening when the father returned from work he noticed the boy looked very ill and called a physician, who made a diagnosis of epidemic cerebrospinal meningitis and gave 15 c.c. of serum intraspinously. Patient did not complain of pain, and fever was not observed until shortly before death. There was a petechial rash scattered over the body. A spinal fluid examination from a puncture done on the evening of the 18th showed a very slightly cloudy fluid. The Nonne and cell count were negative and no organisms were found in smears or cultures (the spinal fluid examinations in these cases were made forty-eight to sixty hours after the spinal puncture, having been sent through the mail to Minneapolis). The patient died at 10.00 A.M., December 19, after an illness of only twenty-four hours.

Autopsy was performed December 20, 1919, twenty-nine hours after death. The body was that of a well-developed and well-nourished boy. There was no edema or jaundice. Scattered hemorrhages varying from the size of a pinhead to a diameter of 3 cm. were present in the skin of the trunk, limbs and face. Examination was permitted for the head only.

The meninges over the convex surface of the cerebrum showed moderate edema with a small amount of thick greenish purulent exudate in some of the sulci. There was a small amount of glairy semisolid material (not purulent) in the posterior horns of the lateral ventricles. Careful coronal sectioning of the brain showed no gross lesions within the brain substance. Smears of the meningeal exudate showed a predominance of polymorphonuclear leukocytes. Some cells showed what suggested intracellular gram-negative diplococci but in no instance were the organisms absolutely definitely demonstrated. Cultures from the meninges and lateral ventricles remained sterile.

Sections of the cortex extending for over 1 cm. into the cerebrum showed microscopically congestion of the vessels with perivascular lymphocytic infiltration. There was a proliferation of glial tissue and a diffuse lymphocytic infiltration. The neurone cells showed chromatolysis with invasion of lymphocytes (satellitosis and neuronophagia). These changes were found in the parts of the sections farthest from the surface of the brain.

The autopsies on this and the previous case were done on the same day. At that time it seemed probable to the pathologist

that he was dealing with cases of epidemic cerebrospinal meningitis; the first case had shown an abundant greenish thick purulent meningeal exudate with no sign of gross lesions in the brain substance. The smaller amount of the purulent meningeal exudate in the second case was thought to be due to the shorter duration of the illness, with death presumably in the stage of general bacteremia and just as the infection was beginning to localize in the meninges. Examinations of smears from the meningeal exudate were quite suggestive also. The encephalitis was proved only when the microscopic sections were examined. The lymphocytic character of the perivascular and diffuse infiltration; the depth to which the affection extended; the degree of the neuron cell changes all serve to differentiate this encephalitis from the shallow hemorrhagic encephalitis with polymorphonuclear leukocytic infiltration sometimes associated with the meningococcic meningitis.

CASE VI.—R. D. (sister of patient No. X and aunt of patients Nos. V and VIII and living with them), female, aged nine years, came home December 18 with stomachache and sat about the house during the day without expressing desires or complaining. Later there was headache, vomiting and general irritability. Serum treatment was started and continued through the illness. There was a positive Kernig the following day and the purpuric spots and herpes were present. Vision was lost in one eye on the 24th. Rigidity was noted as being less and hearing not so acute on December 25. Parotitis developed on the 29th. The attending physician considered the possibility of the condition being lethargic encephalitis. During the latter part of her illness she slept most of the time but her mind was clear when awake. The temperature went as high as 102° to 103° until January 2. Two spinal fluid examinations were made. December 20 the fluid was clear, Nonne and cell count negative and no organisms were present in smears or cultures. December 25: clear fluid, Nonne trace, cell count negative, no organisms in smears or cultures.

CASE VII.—J. G., male, aged twenty-nine years, living with patients Nos. II and IX and working next door to patient No. 1, complained of general weakness on December 19. He had headaches, bowel disturbances and pain in the abdomen. No Kernig or rigidity was noted. He returned to work the following day and was considered as a possible abortive case.

CASE VIII.—M. K., female, aged thirteen years, living with patients Nos. V, VI and X, became ill about 11.00 A.M., December 21. On that date she complained of a mild headache and some pain in the back. The temperature was 102.5° and the pulse 120. She had a tendency to a positive Kernig and there was some pain

upon bending the neck. By the 24th she was apparently well. Spinal fluid examination on the 22d showed a clear fluid with a negative Nonne and normal cell count. No organisms were found in smears or cultures.

CASE IX.—C. G., female, aged twenty-five years, living with patients Nos. II and VII, began to have headache and vomited on December 24. There was some muscular rigidity after the third day and the Kernig sign was positive early in the course of the disease. Deafness developed on the 25th and blindness in the left eye on the 26th. There was general hyperesthesia from the onset of the illness. An erythematous eruption appeared on the 27th. She would talk rationally when someone was conversing with her but otherwise talked more or less irrationally. Headache was severe and persistent from the first. Hearing gradually returned and when last seen on January 11th, the patient was apparently slowly recovering. A spinal fluid examination made December 27th showed a slightly cloudy blood tinged fluid with a normal cell count and no organisms present in the smears. A Gram-positive organism was found in the broth cultures, but there was no growth on serum. On December 28 the fluid was cloudy, pus cells were present and staphylococci were found in smears and in broth and serum. The fluid examination on the 29th gave a cloudy fluid, flocculent. Pus cells were present but no organisms were found in smears or cultures.

CASE X.—R. D., male, aged twelve years, became ill on December 30. At the time of onset he was living with patients Nos. V, VI and VIII. His past history was negative except that he had had anterior poliomyelitis in 1916. His illness began with restlessness and pain in the distribution of the left supra-orbital nerve. On the 31st he complained of tenderness over the left eye. There was no rigidity or tremor. The pulse was 78 and the temperature 98.4°. On the second day of his illness there developed deep and rapid respiration with a respiratory rate of 40 to 60 a minute. There was occasional vomiting. The blood examination at this time showed a leukocyte blood count of 17,000. On January 1 he was able to walk to an automobile, go to another house nine miles away from the village, and walk upstairs to bed. The blood examination on January 4 gave a leukocyte count of 7600 and a red blood cell count of 4,000,000. On this date, there was some cough and expectoration, and percussion gave a higher pitched note over the right upper lobe. Temperature was 101° and the pulse 90. He became semidelirious on the 5th. He answered questions readily and there was no difficulty in his understanding what was said to him. His condition became worse on the seventh but there was no Kernig present and only slight rigidity on extreme

flexion of the neck. In the following few days the mild delirium continued. He would pick at imaginary objects in the air but

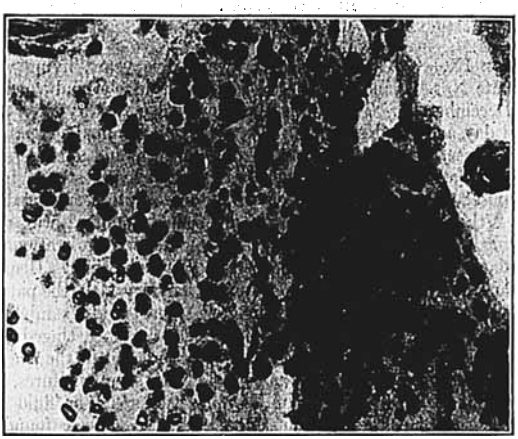

FIG. 1.—Case X. Meninges over cerebral cortex. Arachnoid infiltrated with polymorphonuclears, lymphocytes and endothelial leukocytes. Some extravascular red blood cells also shown.

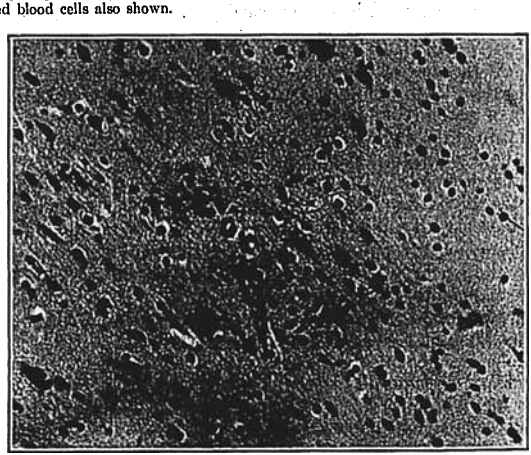

FIG. 2.—Case X. Subcortical white matter of cerebrum. Diffuse lymphocytic infiltration and glial proliferation.

memory and comprehension were unimpaired and in conversation he appeared normal. A neurological examination made on January 9 gave the following findings: Vision was subjectively fair; he saw objects larger or smaller at times. The fundi were normal

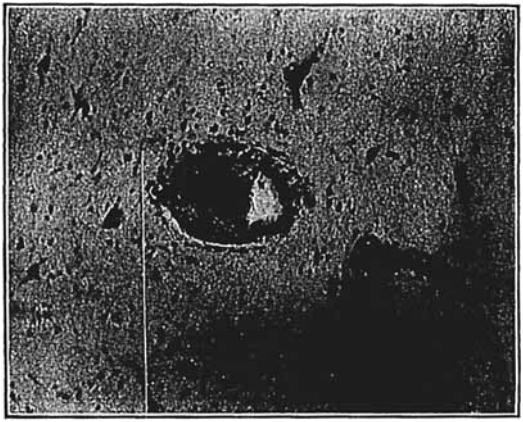

FIG. 3.—Case X. Midbrain. Perivascular lymphocytic infiltration.

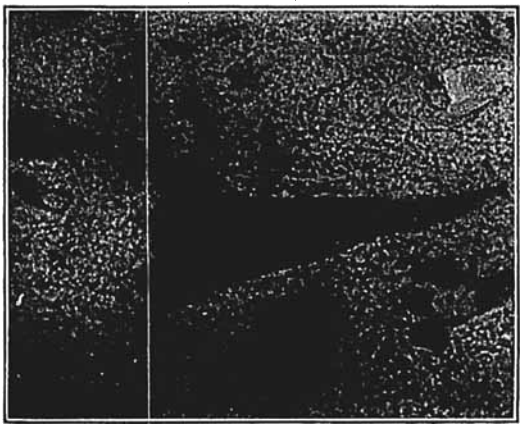

FIG. 4.—Case X. Region of cortex. Satellitosis and chromatolysis of neurone cell.

except for a very slight edema of the disks. There was weakness of the right lateral rectus and a slight ptosis. The pupils were unequal but reacted normally to light and distance. There was

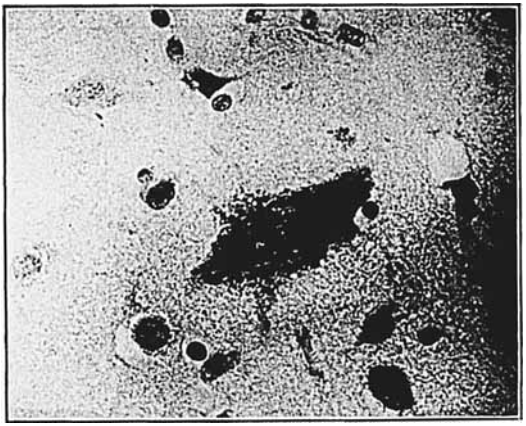

FIG. 5.—Case X. Region of cortex. Disintegration of neurone cell; also shows neuronophagia beginning.

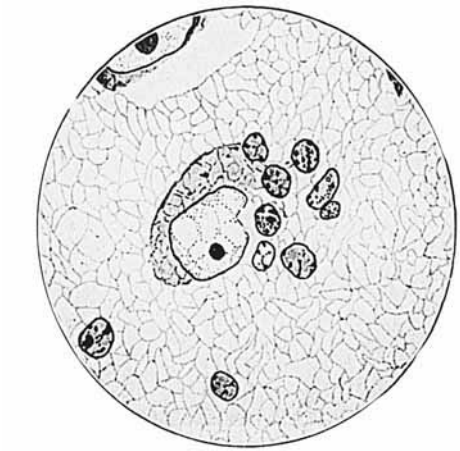

FIG. 6.—Case X. Basal ganglia. Advanced neuronophagia.

no diplopia; convergence was good. On looking to the right a very marked nystagmus was present. A twitching of the facial muscles was noted. Hearing was normal. Swallowing was unimpaired. The relatives thought that there was some change in his voice. He did not ordinarily complain of pain, but his sister said he complained of pain when bathed and of pain at times in the knees. Pressure over the extremities or stroking did not cause discomfort.

There was no atrophy present except in the left leg (poliomyelitis in 1916). General motor and psychic restlessness was evident. He constantly tossed about and had a coarse jerking of the arms and legs. In a general way this resembled choreic restlessness,

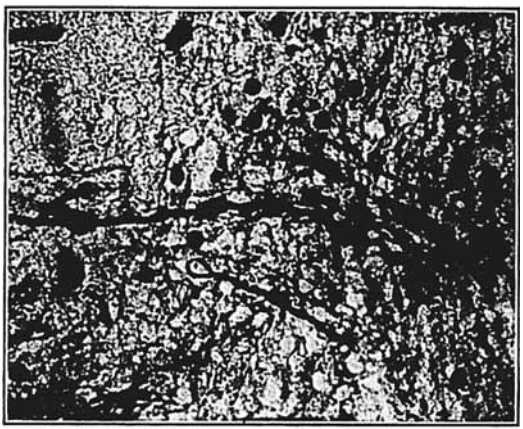

FIG. 7.—Case X. Internal capsule in region of thalamus. Myelin sheath degeneration; showing bulbous enlargements and irregular thickening of the sheaths.

but the movements were coarser and more purposeful although involuntary. A degree of ataxia was noted in the movements of the hands. The deep reflexes were all moderately active except in the left leg where the knee-jerk was absent and the ankle-jerk was much diminished. The plantar reflexes were both flexor in type and there was no ankle clonus. The abdominal reflexes were present. Memory and comprehension were good. He answered questions readily and intelligently. When not in conversation he sang and hummed to himself and also muttered some. He complained of feeling tired and drowsy and went to sleep at times while eating. Spinal fluid examination made January 8 showed a clear fluid, Nonne negative, normal cell count and no organisms

in the smears. Gram-positive staphylococci were present in the cultures. He died January 23, 1920, forty-one days after the onset of his illness.

At autopsy, January 25, 1920; thirty-three hours postmortem, the body was well developed and well nourished. There was no edema or jaundice and the pupils were regular and equal. The lips showed sordes. The serous cavities showed no adhesions and no excess fluid. On the anterior surface of the heart in the epicardium were about a dozen petechial hemorrhages, especially over the right ventricle. There were no other gross lesions in the heart aside from a slight hypertrophy of the left ventricles. The lungs showed no lesions aside from edema in the lower lobes. The spleen was enlarged and very soft and flabby. It showed acute splenitis both grossly and microscopically. The kidneys showed cloudy swelling. On the outer surface of the skull there were a few small hemorrhages. The upper surface of the cerebrum showed a considerable collection of serous fluid which was very slightly cloudy. On the inferior surface of the frontal and parietal lobes and over all the surface of the cerebellum, midbrain, pons, medulla and cervical portion of spinal cord there was congestion of the meningeal vessels with many small hemorrhages. Over these same areas there was a thick fibrinous exudate and in the sylvian fissure this exudate was fibrinopurulent in spots, causing obliteration of the fissure by adhesion. There was about 2.5 c.c. of clear fluid in each lateral ventricle. There was a small amount of thin serous exudate in both middle ears and mastoid cells. The mucous membranes of the middle ears were thickened. The frontal, ethmoid and sphenoid sinuses were normal.

On microscopic examination the various gross findings in the organs of the thorax and abdomen were corroborated. The brain, however, showed significant lesions. In the meninges there was a marked exudate in the subarachnoid space in addition to the edema and hemorrhages noted grossly. In the exudate medium-sized mononuclear leukocytes predominated. There were also numerous polymorphonuclear leukocytes and some lymphocytes. Brain substance: there was a perivascular lymphocytic infiltration, especially marked in the region of the basal nuclei and red nucleus, and somewhat in the cerebellum. Satellitosis, neuronophagia, chromatolysis and further neurone cell degenerations were found especially in the basal nuclei and pons. There was a very marked irregular swelling of the medullary nerve sheaths in the midbrain and pons with irregular thickenings of these sheaths. Small amounts of brown pigment were noted in some cells in the pons not far below the locus cæruleus. At least one clump of small bacilli was found in a stained section of the pons but no further bacteria could be demonstrated even in sections stained for both Gram-negative and

Gram-positive organisms. Cultures from the meninges, ventricles and brain substances were sterile.

Small bits of brain tissue removed from cortex, midbrain and pons, under aseptic precautions, were taken to the Division of Preventable Diseases of the State Board of Health and there inoculated intradurally into rabbits.

One rabbit died approximately forty-eight hours after inoculation. The other rabbits remained alive and apparently well. At autopsy the rabbit which died showed a very heavy fibrinous exudate over the lower lobe of the right lung and congestion of the organs, but no other gross lesions. A very small Gram-negative bacillus growing well on ordinary media was demonstrated in both pleura and heart's blood. This condition may possibly, though not necessarily, have been present before the inoculation. The microscopic examination of the brain was nevertheless significant and it seems entirely possible that the condition found there was related to the inoculation rather than to the pleuritis and bacteremia. In the meninges there was congestion with slight hemorrhage and rather slight diffuse exudate consisting principally of lymphocytes and plasma cells. The midbrain showed a marked perivascular infiltration of lymphocytes and diffuse lymphocytic infiltration and various degrees of degeneration of the large nerve cells with chromatolysis, satellitosis and neuronophagia. Perivascular lymphocytic infiltration was found also in the cerebrum and pons.

CASE XI.—After our last visit to the village, another case has developed in the family (X) and within six days after his death, and, therefore, related to patients Nos. VI, VII, VIII and X. This patient, T. K., aged fifteen years, is said to have had a typical lethargic type of encephalitis. The first symptom was noted January 29, 1920.

DISCUSSION. In these cases we have a rather remarkable sequence. The first cases (I, II and III) presented more or less the picture of an acute toxic condition without special localizing phenomena. In the next group (IV, V, VI, VIII and IX) the meningeal symptoms were prominent with, however, fairly definite evidence of an accompanying encephalitis. The last two cases (X and XI) were primarily encephalitis, with meningeal involvement occurring seven days after the onset of the symptoms in case No. X. In other words there was an infection of an extremely virulent type gradually becoming attenuated and presenting in this course three fairly distinct syndromes—an acute toxemia, a meningitis or meningo-encephalitis and an encephalitis.

In this connection it is interesting to note that in some of the previous epidemics botulism was suspected and a number of cases so diagnosed according to the report of the Local Government

Board (London). In the first cases of our series, botulism was thought of as a not unlikely diagnosis. The infection in these cases was so virulent and the course so acute that few localizing phenomena were present.

It seems likely that these cases are related etiologically to the hitherto reported cases of epidemic encephalitis. Two of these cases presented typical syndromes of "lethargic encephalitis," one of these (No. X) having the choreiform movements seen in a certain number of cases of epidemic encephalitis as well as the characteristic lethargy. In the history of case No. IV we note that the patient stated that she felt well except for the need of a good night's sleep. She was drowsy most of the time but would answer questions rationally.

The autopsies of cases Nos. V and X show lesions typical of epidemic encephalitis.

The perivascular lymphocytic infiltration, most marked in the region of the basal nuclei, the diffuse lymphocytic infiltration with glial proliferation, the degenerative changes in the nerve cells, the satellitosis and neuronophagia, have all been repeatedly described time and again in reports of epidemic encephalitis. The fact that these lesions may be found in the brain cortex, as in our case No. V, has previously been demonstrated in Bassoe's series of cases.

Pathologically, the distinctive feature in this series of cases is the extraordinary prominence of the meningitis. Although meningeal affection has been described by Bassoe and Hala and Smith we have seen no reports of cases showing such an extensive and marked involvement.

The acuteness of the disease process and the mortality of this epidemic are unusual. The mortality was 54.5 per cent. even when case No. VII is included (abortive case). Three of the 6 fatal cases died within approximately twenty-four hours of onset and one other died within forty-eight hours; one after five days and one after forty-one days. Epidemic encephalitis has hitherto been considered generally as a less acute disease with a fairly low mortality.

Besides the acuteness of the disease process and the marked meningeal involvement, these cases are notable for apparent contagiousness. There is a definite relationship between cases Nos. I, II, VII and IX; patients Nos. II, VII and IX lived in one house; patient No. I worked next door to patient No. VII. Patients Nos. III and IV lived in one house and became ill at almost the same time. Patients Nos. V, VI, VIII and X became ill while in the same house and within a period of eight days. During his illness patient No. X was taken to another house; within a short time after his death and in the house where he died, case XI developed the disease. All the 11 cases are included in these three

groups. The first 10 of the 11 cases developed within a period of thirteen days.

	Duration.	Outcome.	Date of onset.	Relationship.
I	21 hours	Died	Dec. 9, 1919	a
II	48 hours	"	Dec. 12, 1919	a
III	24 hours	"	Dec. 12, 1919	b
IV	5 days	"	Dec. 13, 1919	b
V	24 hours	"	Dec. 18, 1919	c
VI	7 days	Lived	Dec. 18, 1919	c
VII	1 day	"	Dec. 19, 1919	a
VIII	3 days	"	Dec. 21, 1919	c
IX	?	"	Dec. 24, 1919	a
X	41 days	Died	Dec. 30, 1919	c
XI	?	?	Jan. 23, 1920	c

Summary. This paper contains a report of 11 cases divided into three groups according to the symptomatology. The first group presented the syndrome of an acute toxic condition; the second group that of a meningitis or meningo-encephalitis and the third group the clinical and pathological findings of epidemic encephalitis. In these cases there was a definite diminution of acuteness in the latter cases and there was an evident contagiousness present in the whole group of cases.

Note. We wish to express our appreciation to Doctors Metcalf, McCrae and Stanley of Fulda, Minnesota, for the courtesy of allowing us to use their clinical records of these cases; to the members of the Division of Communicable Diseases of the State Board of Health for their kindness in making these cases available to us and for the use of their bacteriological and serological records; to Doctors H. E. Robertson and A. S. Hamilton, Chiefs of the Department of Pathology and Public Health and the Division of Nervous and Mental Disease respectively, to Dr. J. C. McKinley for the drawing and to Mr. Henry Morris for the microphotographs.

ANEURYSM OF THE HEPATIC ARTERY: WITH THE REPORT OF A CASE.*

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Aneurysm of the hepatic artery is a very rare condition, only 54 cases appearing in literature. In 1908, Rolland[1] published a study of 40 cases and at the same time reported a case of his own. In addition the following cases have been recorded and are given in

* Reported by Dr. Thomas McCrae with special reference to the clinical features in the International Clinics for September, 1920.

[1] Glasgow Med. Jour., 1908, lxix, 342-358.